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Diagnostic Performance of Cardiovascular Magnetic Resonance in Patients With Suspected Acute Myocarditis

Comparison of Different Approaches

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OBJECTIVES The aim of this research was to identify the diagnostic performance of gadolinium-enhanced and T2-weighted cardiovascular magnetic resonance (CMR) in suspected acute myocarditis.

BACKGROUND Acute myocarditis is difficult to diagnose; CMR provides various means to visualize myocardial inflammatory changes. A CMR approach with clear-cut diagnostic criteria would be desirable.

METHODS We investigated 25 patients with suspected acute myocarditis (18 males, 44 ± 17 years) and 23 healthy controls (13 males, 29 ± 10 years). Cardiovascular magnetic resonance studies included the following sequences: 1) T2-weighted triple inversion recovery; 2) T1-weighted spin echo before and over 4 min after gadolinium injection; and 3) inversion recovery-gradient echo 10 min after gadolinium injection. Qualitative and quantitative image analysis was performed for: 1) focal and global T2 signal intensity (SI); 2) myocardial global relative enhancement (gRE); and 3) areas of late gadolinium enhancement (LGE).

RESULTS Both global T2 SI and gRE were higher in patients than in controls (T2: 2.3 ± 0.4 vs. 1.7 ± 0.4; $p < 0.0001$, gRE: 6.8 ± 4.0 vs. 3.7 ± 2.3; $p < 0.001$). The sensitivity, specificity, and diagnostic accuracy for T2 (cutoff value of 1.9) were 84%, 74%, and 79%, respectively; gRE: (cutoff value of 4.0) 80%, 68%, and 74.5% respectively; LGE: 44%, 100%, and 71%, respectively. The best diagnostic performance was obtained when "any-two" of the three sequences were positive in the same patient yielding a 76% sensitivity, 95.5% specificity, and 85% diagnostic accuracy.

CONCLUSIONS A combined CMR approach using T2-weighted imaging, early and late gadolinium enhancement, provides a high diagnostic accuracy and is a useful tool in the diagnosis and assessment of patients with suspected acute myocarditis. (J Am Coll Cardiol 2005;45: 1815–22) © 2005 by the American College of Cardiology Foundation

Identifying patients with acute myocarditis is a challenging task. Clinical presentations often mimic other disorders and may vary from flu-like symptoms or subclinical disease to acute heart failure and sudden cardiac death (1). Of the imaging approaches utilized to diagnose the disease, cardiovascular magnetic resonance (CMR) has emerged as an important tool. The two relevant gadolinium-enhanced CMR approaches described so far depend on the measurement of myocardial global (early) relative enhancement (gRE) (2) or the visualization of late gadolinium enhancement (LGE) (3). Each of these approaches monitors a different aspect of myocardial injury in myocarditis. Whereas gRE likely reflects myocardial hyperemia and increased capillary permeability as features of present inflammation, LGE mostly indicates irreversible myocardial injury. Another interesting and yet inadequately studied noncontrast CMR approach in myocarditis is T2-weighted imaging, which almost exclusively depends on the detection of myocardial edema. It has been shown to be of diagnostic value (4), but experience has been reported only sparsely. The diagnostic performance of these techniques to identify myocarditis is not well-defined. For example, the reported sensitivity of LGE to detect acute myocarditis varies from 44% to 88% (3,5). Furthermore, a comprehensive CMR protocol combining data obtained from each approach has not reached the clinical arena and yet appears promising for two reasons: first, the spectrum of myocardial injury caused by the disease is diverse, ranging from mild inflammation with hyperemia or edema to frank necrosis (6). One would then expect that an imaging approach designed to detect only one of these injuries would lack sufficient sensitivity. Second, providing information on the various myocarditis-induced injuries could help identify patients with a severe form of the disease or those with a potentially unfavorable prognosis.

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METHODS

Patients. Inclusion criteria:

1) Symptoms and signs suggestive of cardiac disease (angina pectoris, dyspnea, palpitations).

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Abbreviations and Acronyms

CK = creatine kinase
CMR = cardiovascular magnetic resonance
gRE = global relative enhancement
LGE = late gadolinium enhancement
SI = signal intensity

2) Evidence for myocardial injury as defined by electrocardiogram (ECG) changes (ST-segment changes, conduction defects) and elevated serum markers (creatine kinase [CK], troponin T or I).
3) Exclusion of coronary artery disease by angiographic and/or clinical criteria.

Criteria of exclusion were previous myocardial infarction, evidence of chronic myocarditis, and known contraindications to CMR.

Control group. Twenty-three healthy volunteers (13 males, age 29.3 ± 10 years) with no current or past evidence of cardiovascular disorders served as our control group.

A written informed consent was obtained from each subject, and the local ethics committee approved the study.

CMR. Cardiovascular magnetic resonance studies were performed in a 1.5-T system (Signa CV/*i*, GE Medical Systems, Milwaukee, Wisconsin). Localization was performed using breath-hold real time and steady-state free precession images of true anatomical axes of the heart. For the T2- and T1-weighted spin echo sequences, which were used for a quantitative evaluation, the body coil was used. We applied a breath-hold, black-blood, T2-weighted, triple inversion recovery sequence (TR 2 × RR, TE 65 ms, TI 140 ms) in three (basal, midventricular, and apical) short-axis slices (slice thickness 15 mm, gap 5 mm, field of vision 34 to 38 cm, matrix: 256 × 256). Breath-hold steady-state free precession images (TR 3.8 ms, TE 1.6 ms) were acquired in two- and four-chamber views to assess global ventricular function. We then applied a free breathing spin echo sequence in four identical axial slices both before and after (without any change in parameters in between) intravascular injection of 0.1 mmol gadolinium-diethylenetriaminepentaacetate (DTPA) (Magnevist, Schering, Germany) using an automated injector (Medrad, Indianola, Pennsylvania). The sequence was started immediately after injection and lasted 3 to 4 min; thus, the images reflect gadolinium enhancement at a mean of 2 min. After the acquisition of spin echo images, an additional dose (0.1 mmol) of gadolinium-DTPA was injected, and a breath-hold contrast-enhanced inversion-recovery gradient-echo sequence (TR 5.5 ms, TE 1.4 ms, TI 225 to 275 ms as individually optimized to null myocardial signal, matrix 256 × 192, slice thickness/gap 15/5 mm) was applied after a delay of 10 min in three short- and three long- (two-, three-, and four-chamber views, respectively) axis slices.

Coronary angiography. Coronary angiography was performed on a standard angiography suite (Hicor, Siemens, Erlangen, Germany) in 21 patients to exclude the presence of significant coronary artery disease (>70% stenosis).

Clinical analysis. Two observers (A.Z. and P.B.), who were blinded to CMR data, assessed the clinical course of the patients during their hospital stay.

Image analysis. SPIN ECHO IMAGES. Regions of interest covering the left ventricular myocardium as well as within a skeletal muscle (erector spinae or lattisimus dorsi) in the same slice were manually drawn in the precontrast images and were copied to the postcontrast images (Fig. 1), and gRE was calculated as previously described (2).

T2-WEIGHTED IMAGES. Quantitative analysis: regions of interest were drawn covering the left ventricular myocardium and within a skeletal muscle in the same slice. The myocardial signal intensity (SI) was related to that of the skeletal muscle:

$$\text{Relative myocardial T2 SI} = \text{SI}_{\text{myocardium}} / \text{SI}_{\text{skeletal muscle}}$$

Endocardial and epicardial contours were manually drawn, and focal areas of high T2 SI (those with SI more than the normal myocardium plus two standard deviations) were identified (MASS 6, Medis, Leiden, the Netherlands).

QUALITATIVE ANALYSIS. This was performed by the consensus agreement of two observers (J.S-M. and H.A-A.) who were blinded to the patients' clinical data. Images were evaluated for the presence or absence of focal or segmental areas of high T2 SI.

LGE. QUALITATIVE ANALYSIS. This was done for the presence, number, and transmurality of LGE areas.

QUANTITATIVE ANALYSIS. Areas of LGE (those with SI more than the normal myocardium plus two standard deviations) were delineated similar to that in T2 imaging. Regions of interest were also drawn within background air. The contrast-to-noise ratio (CNR) and the signal-to-noise ratio (SNR) of LGE were then calculated as follows:

$$\text{CNR} = (\text{SI}_{\text{LGE}} - \text{SI}_{\text{myocardium}}) / \text{SI}_{\text{noise}}$$

$$\text{SNR} = \text{SI}_{\text{LGE}} / \text{SI}_{\text{noise}}$$

Foci of high signal in delayed enhancement and T2 images were traced, and their volume expressed as a percentage of the total myocardial slice volume.

Statistics. All statistical tests were performed using a commercially available statistical program (SPSS 11 for Macintosh, SPSS Gmbh Software, Munich, Germany). Data are presented as mean ± SD. Continuous variables were compared using the Mann-Whitney *U* test and noncontinuous data using the chi-square test. Data were correlated using the Spearman correlation coeffecient. Reciever operating charcteristic curves were used to identify the cutoff values of gRE and global T2 signal changes. A p value <0.05 was considered significant.

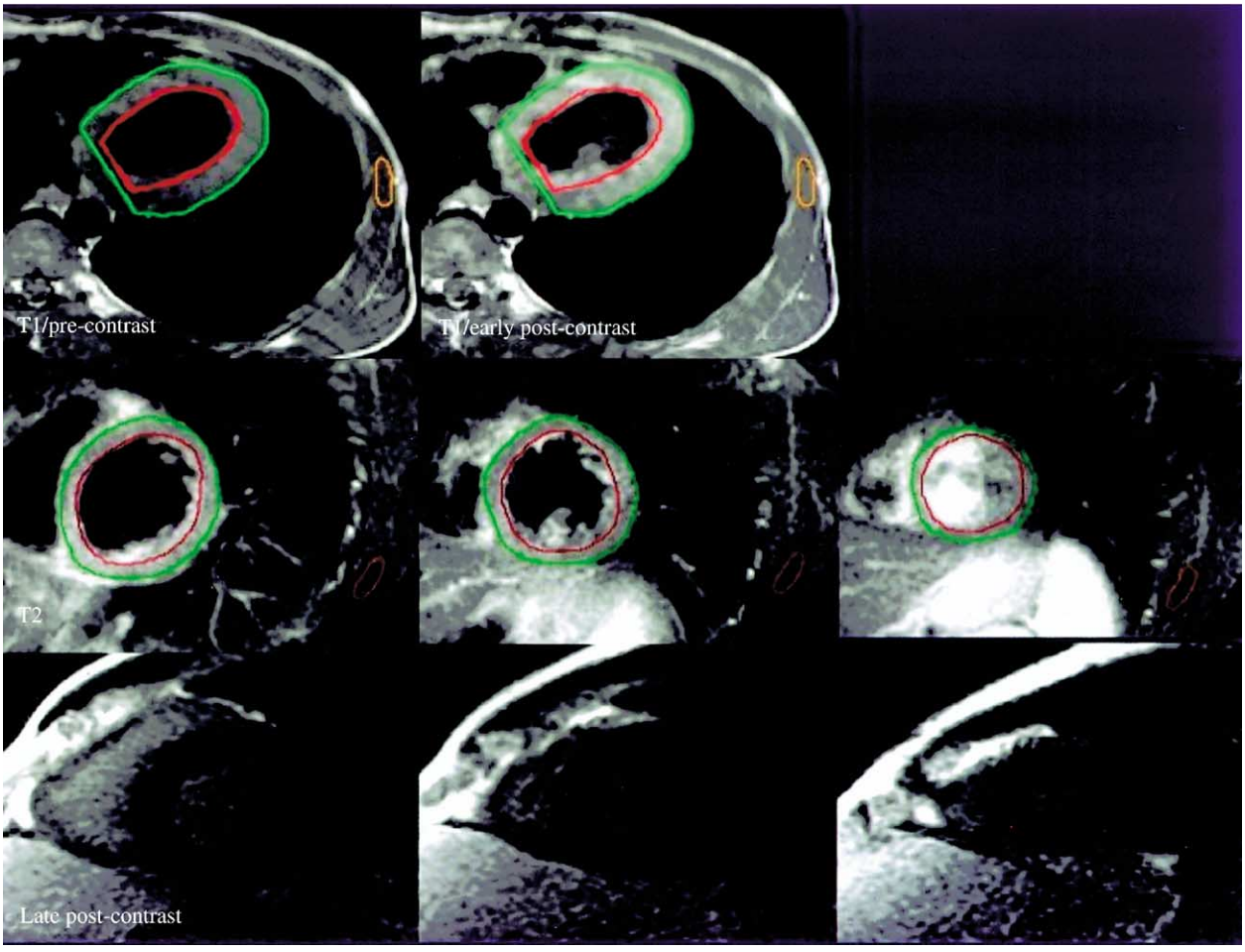

Figure 1. Cardiovascular magnetic resonance images from Patient #6. **(Top)** Pre- and postcontrast axial T1-weighted spin echo images of the same slice. Global relative enhancement was elevated (4.6). Regions of interest are drawn around the myocardium and within the skeletal muscle. **(Middle)** Short-axis T2-weighted images: no focal areas of high T2 signal, yet the higher global myocardial T2 signal in relation to the skeletal muscle is visibly appreciable (exact value = 2.5). **(Bottom)** Corresponding late enhancement images: no evidence of late gadolinium enhancement.

RESULTS

Table 1 provides the characteristics of the study patient population. The average duration between the onset of cardiac symptoms and CMR was 5.6 ± 4.2 days. Patients had significantly lower ejection fraction than controls (57.1 ± 12.6% vs. 64.2 ± 5.2%, p = 0.013). Biopsy was performed in two patients (Patients #5 and #25) and showed acute giant cell myocarditis with round cell infiltration and multiple necrotic foci in Patient #5 and diffuse fibrosis, myocardial hypertrophy, regional inflammatory cell infiltration, and fresh erythrocyte extravasation in Patient #25.

gRE. Global relative enhancement was significantly higher in patients compared to controls (6.8 ± 4.0 vs. 3.7 ± 2.3; p < 0.001). Figures 1 and 2 show representative images from two patients with increased gRE. Figure 3 shows the receiver operating characteristic curve of gRE to identify myocarditis. A cutoff value of 4.0 had a sensitivity, specificity, and diagnostic accuracy of 80%, 68%, and 74.5%, respectively; gRE did not significantly correlate with ejection fraction (p = 0.350) or hospital stay (p = 0.320).

T2-weighted imaging. The global myocardial T2 SI ratio was significantly higher in patients than in controls (2.3 ± 0.4 vs. 1.7 ± 0.4; p < 0.0001). Figure 3 shows the receiver operating characteristic curve of global myocardial T2 SI to identify myocarditis. A cutoff value of 1.9 had a sensitivity of 84%, specificity of 74%, and a diagnostic accuracy of 79%; T2-SI ratio was not related to gRE (p = 0.462). Moderate (0.42) significant correlation was found between T2-SI ratio and troponin levels (p = 0.035). Eight patients showed focal areas of high T2 SI, which were either transmural or subepicardial (Fig. 4) but never confined to the subendocardium. These eight patients had significantly higher peak CK (781.6 ± 695.4 vs. 254.5 ± 173.1; p = 0.014). Corresponding areas of LGE were noted in seven of these eight patients with a spatial extent significantly smaller than areas of high T2 SI (13.8 ± 8.2% vs. 21.6 ± 5.8%; p = 0.018).

LGE. The sensitivity, specificity, and diagnostic accuracy of LGE were 44%, 100%, and 71%, respectively. Posterolateral and inferior segments were most likely to be affected (73%) followed by anterior (36%) and septal (27%) seg-

Table 1. Characteristics of Myocarditis Patients

Patient #	Age (yrs)	Gender	Symptoms	EF	ECG	CA	CK*	TnT/I†	gRE	T2	LGE
1	38	M	ACP	56	ST-segment elevation: II, III, aVF T-negative: I, II	yes	2,267	3.6	3.8	2.0	yes
2	50	F	ACP	47	T-negative: I, II, aVL, aVF, V_2–V_6	yes	51	1.3	2.8	2.5	no
3	35	M	ACP	70	ST-segment elevation: I, II, aVF, V_5, V_6	yes	166	0.2	2.3	2.7	no
4	63	F	ACP, D	49	ST-segment elevation: I, II, aVL; T-negative: III	yes	429	70.1	10.1	2.3	no
5	39	F	D	26	ST-segment elevation: V_2–V_4 ST-segment depression: V_5, V_6 T-negative: I, aVL, V_1–V_3	yes	246	274	11.0	3.3	yes
6	26	M	ACP	65	ST-segment elevation: I, II, aVF, V_4–V_6	np	8.5	2.0	4.6	2.5	no
7	36	F	D, ACP	51	T-negative: III, aVF	yes	407	8.5	2.5	2.2	no
8	18	M	ACP	61	ST-segment elevation: II, III, aVF, V_5–V_6 T-negative: II, III, aVF, V_5–V_6	yes	168	41.0	4.4	3.0	yes
9	39	M	ACP	66	ST-segment elevation: V_4–V_6	yes	292	29.0	9.3	1.6	yes
10	38	M	ACP	63	ST-segment elevation: II, III, aVF, V_5–V_6	yes	213	15.7	9.0	2.2	no
11	37	M	ACP	63	ST-segment elevation: I, II, aVL, V_4–V_6	yes	1,100	103	5.1	2.9	yes
12	50	M	ACP, D	56	ST-segment elevation: I, II, aVF, V_5–V_6	yes	609	70.0	2.8	2.9	yes
13	66	F	ACP	63	T-negative: I, II, aVL, V_2–V_6	yes	547	1.8	4.4	2.2	no
14	29	M	P	42	Atrial fibrillation and J-point elevation: I, II, aVF, V_2–V_6	np	84	0.4	21.0	2.0	yes
15	17	M	ACP	62	ST-segment elevation: II, III, aVF, V_3–V_5 T-negative: I, aVL	np	443	39.0	9.3	2.0	no
16	23	M	P	57	ST-segment elevation: I, II, aVF T-negative: III	np	570	0.3	4.8	2.0	no
17	52	M	ACP	65	ST-segment elevation: V_1–V_6, II, III, aVF	yes	1,023	0.7	8.7	2.1	yes
18	84	F	ACP	74	T-negative: I, II, aVL, aVF, V_2–V_6 ST-segment elevation: V_1–V_2	yes	166	4.8	7.0	2.3	no
19	67	M	ACP, D	76	ST-segment elevation: II, III, aVF, V_4–V_6	yes	213	0.4	6.1	1.7	no
20	44	M	D	45	T-negative: II, III, aVF, V_5–V_6	yes	105	0.1	4.5	1.7	no
21	37	M	ACP	62	ST-segment elevation: II, III, aVF T-negative: II, III, aVF, V_5–V_6	yes	200	1.4	4.1	1.8	no
22	51	M	ACP	67	ST-segment elevation: V_2–V_5 T-negative: III	yes	349	1.6	7.9	2.1	yes
23	40	M	ACP	62	ST-segment elevation: I, II, aVL, V_5–V_6 T-negative: III	yes	594	1.0	10.0	2.2	yes
24	52	M	ACP	54	ST-segment elevation: II, III, aVF, V_2–V_6	yes	20	1.0	5.3	2.3	yes
25	67	F	ACP, D, P	26	Atrioventricular block III	yes	232	3.9	8.9	2.7	no

*Upper limit: 180 I/U; †Upper limit: <0.1 for TnT and <0.3 for TnI.
ACP = acute chest pain; CA = coronary angiography excluding significant coronary artery disease; CK = creatine kinase; D = dyspnea; EF = ejection fraction; gRE = global relative enhancement; LGE = late gadolinium enhancement; np = not performed; P = palpitation.

ments. One patient had LGE involving most of the right ventricular free wall as well. The number of these foci ranged from one to three (>1 in 64%); LGE was always located in the epicardial or midportion of the ventricular wall but never within the subendocardium (Fig. 4). Signal-to-noise ratio and contrast-to-noise ratio (in relation to normal myocardium) were 5.2 ± 2.3 and 3.5 ± 1.7, respectively. There was no significant difference between patients with and those without LGE regarding age ($p = 0.805$), time from onset to CMR ($p = 0.579$), ejection fraction ($p = 0.742$), duration of hospital stay ($p = 0.977$), peak CK ($p = 0.154$), troponin levels ($p = 0.262$), or ST-segment elevation ($p = 0.122$).

Combined approach. The best diagnostic performance was obtained when any two of the criteria obtained by the three techniques were positive (T2: SI ratio 1.9; gRE: SI ratio 4.0; LGE: presence of visually detectable bright areas) in the same patient. This approach had 76% sensitivity, 95.5% specificity, and 85% diagnostic accuracy (Fig. 5). Specifically, gRE and T2 were positive in 64%, LGE and T2 in 40%, LGE and gRE in 36%. The three sequences were all positive in 32% of the patients and in none of the controls.

DISCUSSION

gRE. In agreement with previous reports (2,7), we found that myocarditis patients have an increased gRE. Tissue hyperemia is an integral component of the acute inflammatory reaction of the myocardium, which may explain this finding. Diffuse myocyte injury can also increase the volume of distribution and subsequently the extraction fraction of extracellular compounds like gadolinium-DTPA, resulting in abnormal myocardial enhancement. This concept is supported by the results of Almenar et al. (8) who found a significant correlation between gRE and the presence of

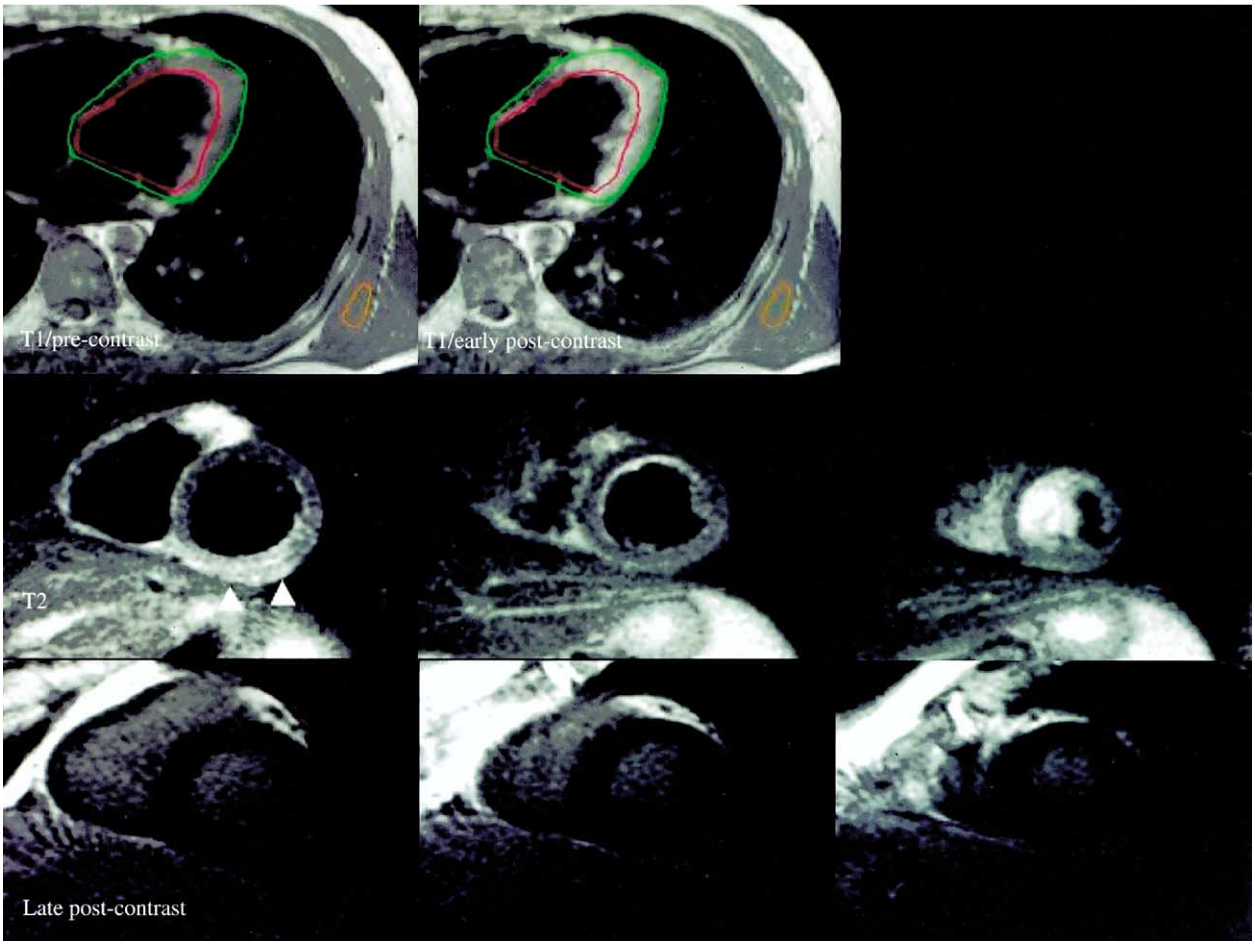

Figure 2. Cardiovascular magnetic resonance findings in Patient #21. **(Top)** Pre- and postcontrast axial T1-weighted spin echo images of the same slice. Global relative enhancement was elevated (4.1). **(Middle)** T2-weighted images in three short-axis slices. Note the posterolateral focal high T2 signal **(arrowheads)** in the basal slice with apparent focal increase in myocardial thickness. **(Bottom)** Corresponding late enhancement images: no evidence of late gadolinium enhancement.

myocyte injury after heart transplantation. The diagnostic performance of gRE to detect myocarditis in our series was lower than that previously reported (7). Two reasons may explain this finding: first gRE measurements depend on the assumption that the skeletal muscles exhibit a "normal" pattern of gadolinium enhancement. This assumption may not hold true in some cases when the inflammatory process extends to involve skeletal muscles as well (9). In such a case, gRE will be "pseudonormalized" even in the presence of abnormal myocardial enhancement. Indeed, patients with negative gRE showed abnormally increased skeletal muscle enhancement (22%). Second, early in the course of myocarditis, the inflammatory process is predominantly focal (10), which could result in a negative gRE. The fact that our patients were studied at an average of six days after the onset of cardiac symptoms supports this notion.

LGE. The exact pathophysiological grounds of LGE in myocarditis are still under investigation. Myocardial necrosis in the acute phase appears to play a major role, but also severe edema could sufficiently increase the volume of distribution of gadolinium to cause visually detectable SI changes. The absence of a significant correlation between LGE and troponin release is not surprising and may reflect one of two possibilities: first, it could be that—at least in some patients—these foci represent replacement fibrosis from previous subclinical episodes of myocarditis, which would then result in gadolinium accumulation similar to that in a chronic myocardial scar (11) in the absence of elevated troponin. Second, diffuse myocarditis could result in troponin release (12) without LGE.

The incidence of LGE in myocarditis is a controversial issue. The 44% incidence we observed is in perfect agreement with the 44% found by Rieker et al. (5). Kuhl et al. (13), using antimyosin scintigraphy, observed focal myocardial cell damage in 55%. Mahrholdt et al. (3), however, reported a much higher incidence of LGE (88%). The reason for discrepancy may be related to differences in patient populations or study designs. Whereas we and Rieker et al. (5) studied patients in the acute phase of the disease, Kuhl et al. (13) and Mahrholdt et al. (3) included a significant fraction of patients with "healed" myocarditis. Moreover, the pattern of myocardial injury is influenced by the virus type (14). This could partially explain differences between our results and those of Mahrholdt et al. (3) where

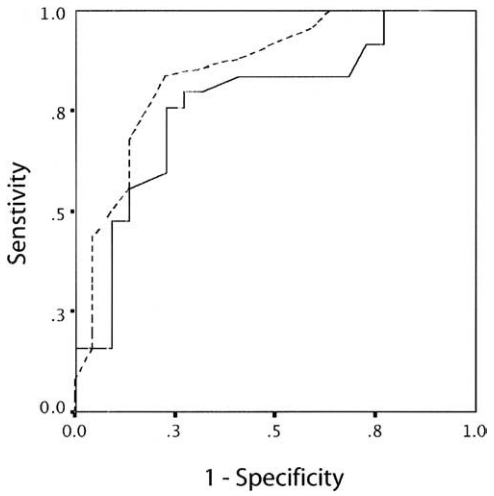

Figure 3. Receiver operating characteristic curves for global relative enhancement **(solid line)** and global relative T2 signal **(broken line)**.

parvovirus was identified as a causative agent in a significant fraction of patients. Parvovirus is unique in selectively injuring the endothelial cells resulting in microinfarcts (15), which may be detectable as LGE.

The classical pathological description of myocarditis, the so-called Dallas criteria (16), can also provide insight into the incidence of LGE in myocarditis. Active myocarditis is defined as inflammatory reaction with myocyte injury. This is expected to result in LGE secondary to the focal expansion of the extracellular space. In borderline myocarditis, however, myocyte injury is lacking, and it is in this group of patients that LGE may not be observed.

Finally, the clinical significance of LGE in myocarditis is yet to be defined. We did not find a significant correlation between LGE and markers of disease severity such as ejection fraction or duration of hospital stay. Nevertheless, the finding that there are two subgroups of myocarditis patients—those with and those without LGE—holds promise that LGE may provide additional significant prognostic information. Specifically, we propose two hypotheses which, if proven to be true, could define an exciting role of CMR to risk-stratify myocarditis patients. First, the link between myocarditis and the later development of dilated cardiomyopathy is well-established (17). Yet only a fraction of myocarditis patients progress to dilated cardiomyopathy. McCrohon et al. (18) found that a group of dilated cardiomyopathy patients exhibit a pattern of focal enhancement similar to the one we observed in myocarditis patients. It seems intriguing to postulate that those myocarditis patients with positive LGE may be more likely to develop dilated cardiomyopathy. Second, the border zone between scar tissue and healthy myocardium is a known substrate for electrical instability. The question of whether myocarditis

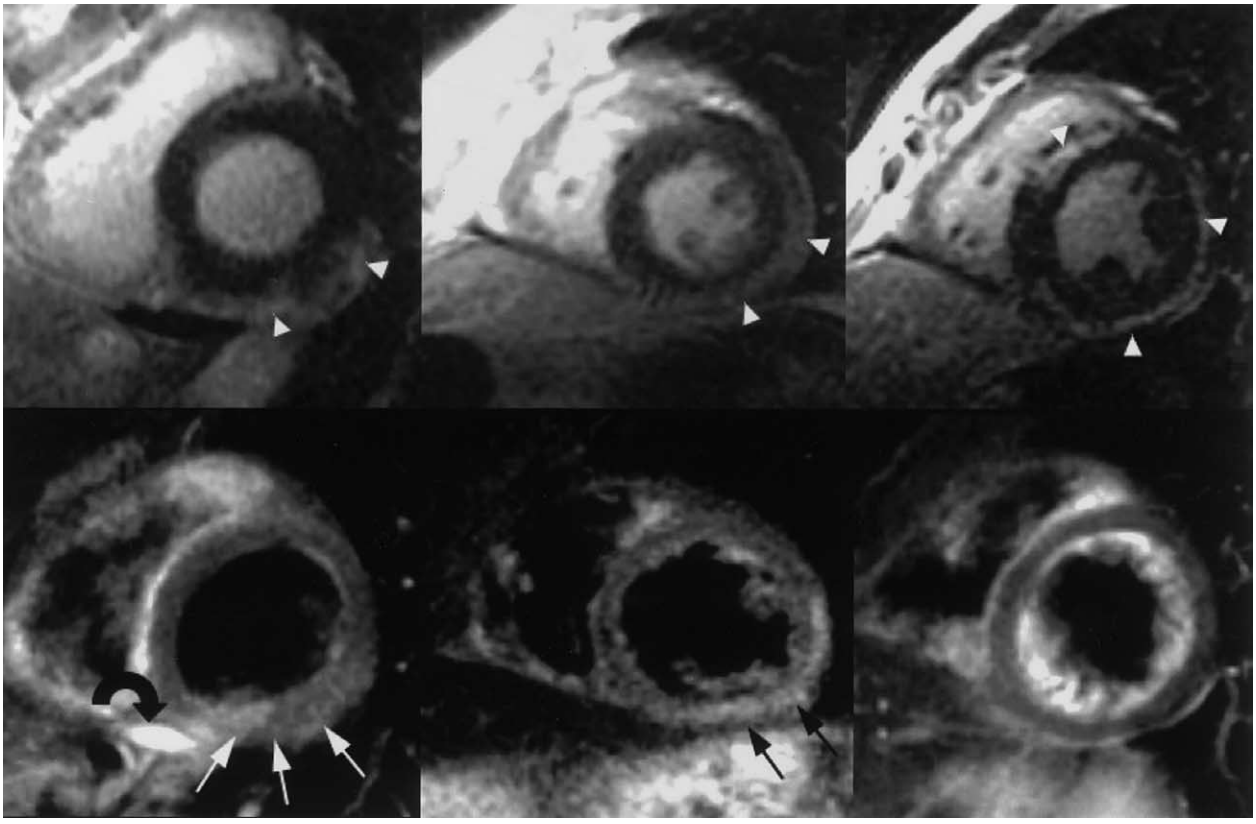

Figure 4. Representative images from 3 patients (#8, #12, and #14 from **left to right**, respectively). **(Top)** Late gadolinium enhancement (LGE). **(Bottom)** T2-weighted. Focal high T2 signal **(thin arrows)** corresponds to areas of LGE **(arrowheads)** in Patients #8 and #12 but not in Patient #14. Note the predominant epicardial distribution of high T2 signal in Patient #12. Small pericardial effusion is seen in Patient #8 **(thick curved arrow)**.

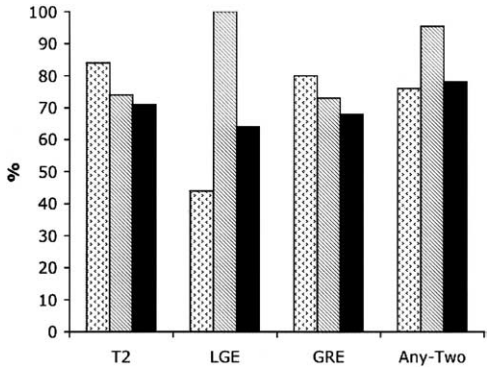

Figure 5. Diagnostic performance of T2, late gadolinium enhancement (LGE), and global relative enhancement (GRE) as compared to the "any-two" approach. **Spotted bars** = sensitivity; **diagonal striped bars** = specificity; **solid bars** = diagnostic accuracy.

patients with LGE would be, thus, more liable to develop ventricular arrhythmias deserves to be a research focus.

T2-weighted imaging. The most likely explanation for the T2 abnormalities we observed in myocarditis patients is the water-sensitive characteristics of this technique, which allows the detection of tissue edema, a substantial feature of the acute inflammatory reaction in the myocardium (19). Other than expected, a focal increase in T2 signal was not always associated with LGE. Although there was a significant correlation between the two findings, many patients had LGE or T2 abnormality only (Figs. 2 and 4). It seems that the evolution/resolution pattern of myocardial edema might be different from that of LGE. Accordingly, at a particular "time window" after the symptoms, only one of the two is detectable. One other possibility would be that focal edema marks a less severe form of myocardial injury, which then may or may not progress to actual necrosis in a cascade similar to that of acute ischemic injury (20). Another unexpected finding was the absence of a significant correlation between the global myocardial T2 signal and global myocardial enhancement. One would expect that tissue edema should increase both myocardial T2 SI as well as the volume of distribution of gadolinium-DTPA with subsequent increase in myocardial enhancement. It could be that global myocardial edema results in a degree of capillary compression hindering abnormal contrast enhancement (21), which then starts to increase with the resolution of edema. This differential time course is supported by the finding that only T2 abnormalities significantly correlated with laboratory markers of acute myocardial injury.

Clinical implications. The "any-two" approach has the potential to increase the diagnostic performance of CMR in the clinical setting as well as in multicenter trials. A significant fraction of acute myocarditis patients present with a clinical picture mimicking that of acute myocardial infarction representing a diagnostic challenge (22,23). Acute myocardial infarction is characterized by focal transmural high T2 signal and subendocardial or transmural LGE (24). This is different from the subepicardial LGE of myocarditis with no focal high T2 signal in the majority of cases.

Study limitations and technical considerations. The parameter that should be used as the "gold standard" to identify myocarditis remains a controversial issue. Some investigators used endomyocardial biopsy to identify the disease (6,22,25), and many others relied instead on a combination of clinical, laboratory, ECG, and angiographic findings (7,23,26,27). We have also relied on this later approach for the following reasons: first, the sensitivity of endomyocardial biopsy to identify myocarditis is limited possibly secondary to the focal nature of the disease (28). Using polymerase chain reaction to identify viral genomes in the myocardium, disagreement with the results of myocardial biopsy was noted in 50% of the cases (29). This likely explains the discrepancy between the low incidence of biopsy-identified myocarditis in many trials and the clinical or postmortem incidence of the disease (25,27,30). Second, the majority of our patients were young with an acute, often fairly unstable presentation; thus, we did not want to subject this group of patients to unnecessary invasive procedures.

Although there is a theoretical possibility that patients in our study suffered from undetectable coronary heart disease, the absence of any coronary stenosis makes an ischemic injury unlikely. In the four patients without catheter verification of the absence of coronary stenosis, neither the risk profile nor other clinical criteria or injury morphology indicated any evidence for coronary heart disease. But, more importantly, the pattern of either a complete lack of scarring or a focal injury distribution not attributable to epicardial coronary artery occlusion makes this very unlikely. Late gadolinium enhancement images were acquired using a slice thickness of 15 mm, which may have reduced the sensitivity to detect small lesions. This was chosen to match the slice thickness of T2 images to maximize the signal-to-noise ratio of this technique. To reduce the possibility of missing small lesions, we acquired additional LGE images in long-axis slices.

Conclusions. A combined CMR approach using T2-weighted imaging, early and LGE provides a high diagnostic accuracy and is a useful tool in the diagnosis and assessment of patients with suspected acute myocarditis.

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